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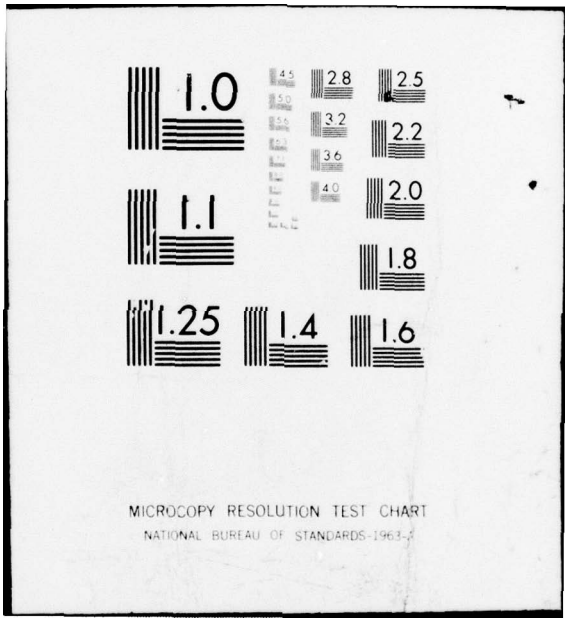
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THE EFFECT OF SELECTIVE AND TOTAL SLEEP LOSS ON THE CNV AND ITS PSYCHOLOGICAL AND PHYSIOLOGICAL CORRELATES

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BUREAU OF MEDICINE AND SURGERY DEPARTMENT OF THE NAVY
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The Effect of Selective and Total Sleep Loss on the CNV and its Psychological and Physiological Correlates[1]

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METHOD

Fourteen Navy enlisted men lived, one at a time, in a sleep laboratory as subjects for 2 weeks. The CNV session, lasting approximately 40 min, was held once in each morning of 4 baseline days (B1, B2, B3 and B4). Following the baseline days, either slow wave sleep (SWS, sleep stage 4), or rapid eye movement sleep (REM) was denied for 3 consecutive nights. After the third night of SWS or REM deprivation, one CNV session (StD3) was held. The sleep stage deprivation was followed by one night of total sleep loss and CNV session (TD). Then, 2 nights of undisturbed recovery sleep with morning sessions (R1 and R2) followed one night of total sleep loss. Of the total of 14 *Ss*, 7 experienced SWS deprivation, and the remaining 7 experienced REM deprivation. The heart rate data to be reported were, however, obtained from only 13 *Ss*: 6 under SWS deprivation, and 7 under REM deprivation.

Each CNV trial started with a presentation of the first stimulus (S_1). The S_1 was either a 0.5 sec "high" (1 kc/sec) or "low" (500 c/sec) tone. Four sec after the S_1, the second stimulus, S_2, a "low" (500 c/sec) tone, was presented to the subjects. With the higher S_1, *Ss* were not required to respond to S_2. With the lower S_1, *Ss* were instructed to terminate S_2 as quickly as possible by closing a hand-held switch, A. The CNV session consisted of two parts: (1) 24 work-paced trials; and (2) 24 self-paced trials. Under the work-paced condition, the experimenter determined the time of each trial presentation. The inter-trial intervals under the work-paced CNV were 20 sec (8 trials), 25 sec (8 trials) and 30 sec (8 trials), all randomly mixed as to presentation. Under the self-paced condition, *Ss* closed a hand-held switch, B, to initiate a CNV trial. By means of an electronic logical network, *Ss*' closure of the switch produced S_1 and subsequently S_2, provided that the switch closure occurred after a lapse of at least 20 sec since the last previous trial. In addition to these CNV trials, S_2 alone was presented to *Ss* 7 times before and after the CNV trials. *Ss* were instructed to turn off S_2 as quickly as possible by closing switch A. Throughout the CNV session, *S* remained in

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bed with eyes open, and kept the eyes fixated on an "X" drawn on cardboard and placed in front of them. Only during the inter-trial intervals was *S* permitted to move the eyes away from the "X" mark to ease eye strain. After each CNV session, *S* completed a post-CNV trial questionnaire while he remained in bed.

The CNV was recorded from the vertex, referenced to linked mastoids. For the CNV recording, time constant of a 9806A AC/DC coupler of a Beckman Dynograph was modified to be approximately 6 sec. The vertical electro-oculogram (EOG) of the right eye was recorded with a similarly modified Beckman coupler. Respiration, electrocardiogram (EKG), skin potential, skin resistance, finger pulse and electromyogram (EMG) of the thumb used for closing hand-held switches were recorded by a Beckman type R Dynograph, using a time constant of 0.3 sec. A Hewlett-Packard 3907C 7-channel instrumentation tape-recorder was used to record vertical EOG, vertex EEG (CNV), EKG, stimulus mark and time code. A calibrator injected a 25 μV pulse to the CNV channel, 2 sec before S_1.

RESULTS

Table I lists the CNV magnitudes, defined as the area of cortical negativity between S_1 and S_2 as measured with a Dietzgen polar planimeter in square millimeters. All *S*s developed stable CNVs under both task-paced and self-paced conditions during the baseline sessions. Both SWS and REM deprivations reduced the CNV magnitudes, but this result achieved significance (at 5% level or better) only under the self-paced condition. No group difference was observed between the REM-deprived and the SWS-deprived subjects, in terms of their CNV magnitudes. In contrast, one night of

TABLE I

MEAN AND ONE STANDARD DEVIATION OF CNV MAGNITUDE, DEFINED AS A TOTAL AREA OF EEG NEGATIVITY DURING THE S_1–S_2 INTERVAL.
($N = 7$.)

	Sleep deprivation	
	REM	SWS
Work-paced trials		
B	17.3 ± 7.6	21.1 ± 7.9
StD3	11.2 ± 8.1	18.6 ± 9.7
TD	11.6 ± 4.2	14.0 ± 6.0
R	18.8 ± 5.9	29.7 ± 8.8
Self-paced trials		
B	23.5 ± 14.6	23.6 ± 15.9
StD3	7.4 ± 4.0	13.9 ± 9.6
TD	8.0 ± 4.6	6.7 ± 7.3
R	19.3 ± 12.2	26.4 ± 14.2

B = Baseline day (average of B3 and B4) session. StD3 = CNV session held after 3 nights of sleep stage deprivation. TD = CNV session held after one night of total deprivation of sleep following StD3. R = Recovery day session.

total sleep loss resulted in a uniform and significant reduction in CNV magnitude, compared with the baseline CNV magnitude, regardless of differences in trial pacings and the particular sleep stage deprivation. However, the CNV was not completely abolished.

Digital computer analysis of averaged heart rate response showed that on the CNV trials (*e.g.*, the discriminant reaction time trials), there was induced a small but reliable heart rate change following S_1, in anticipation of S_2, and after S_2 and the motor response of a switch-closure. Fig. 1 and 2 show the averaged heart rate response, each line representing the means of 13 *Ss*. Fig. 1 shows the averaged heart rate response during the CNV trials under the work-paced condition, and Fig. 2 the same under the self-

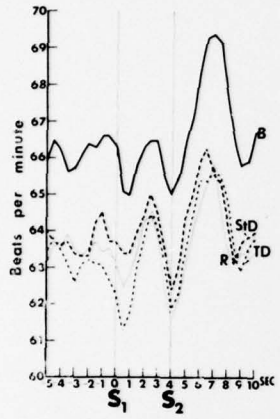

Fig. 1. Averaged heart rate response obtained from 13 subjects under work-paced condition. S_1 = Discriminative warning stimulus of 500 c/sec, 0.5 sec tone. S_2 = Imperative stimulus. The ordinate in heart rate (beats/min), and the abscissa in seconds. B = Baseline. StD = After sleep stage deprivation (REM and SWS deprivations combined). TD = Total sleep deprivation. R = After 1 and 2 nights of recovery sleep.

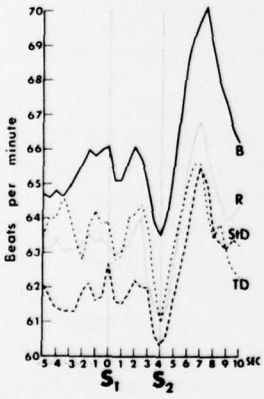

Fig. 2. Averaged heart rate response obtained from 13 subjects under the self-paced (auto-start) condition. See Fig. 1 for abbreviations.

paced condition. The S_1 produced phasic bradycardia which was followed by heart rate increase to or above the pre-S_1 level. The second reliable phasic bradycardia of 2–3 beats/min started approximately 2.5 sec after S_1, and the heart rate reached its minimum at the time of S_2 presentation. A reliable heart rate increase above the pre-S_1 level was observed after S_2 and a hand-held switch closure. Although the group average of heart rate responses was small in terms of absolute change in beats/min as shown in Fig. 1 and 2, the pattern of cardiac changes under the CNV paradigm (*e.g.*, the reaction time paradigm) was clear and large in some subjects, as shown in Fig. 3. In this

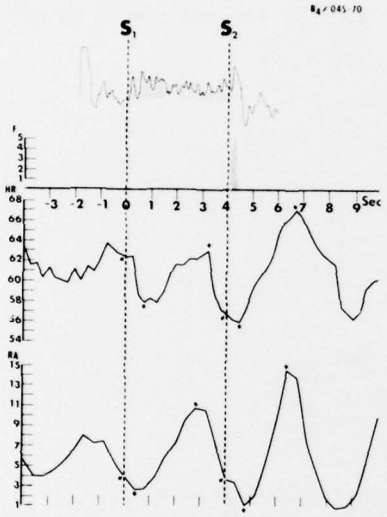

Fig. 3. The CNV, reaction time, averaged heart rate response, and averaged respiratory amplitude from a subject, 04S-70, obtained in his fourth baseline day session. On the top, the CNV is shown with a 25 μV calibration signal. The reaction time is shown, immediately below the CNV plot, as a histogram. Its ordinate is scaled to show a frequency of occurrence of given reaction time. Altogether 10 trials were averaged for the CNV, averaged heart rate and respiratory amplitude responses. Averaged heart rate response is shown below the reaction time histogram. The ordinate, HR, is given in beats/min. The averaged respiratory amplitude response is given last, where the vertical axis represents an arbitrary unit which is, however, proportional to a respiration depth.

illustration a phasic bradycardia just before S_2 was 6 beats/min, followed by a post-S_2 cardiac acceleration of approximately 12 beats/min. This pattern of cardiac responses was not significantly altered by REM, SWS or total sleep deprivation.

There was no consistent skin potential response accompanying the CNV trials.

The detailed analysis of respiratory amplitude response was performed on work-paced CNV data obtained during the B4 and TD sessions. Fig. 3 shows the respiratory amplitude response also from a single subject. The only reliable average respiratory amplitude response was observed with S_2 and the subsequent motor response to turn off S_2: all subjects tended to exhale air shortly after S_2 and to inhale air approximately 2.4 sec after S_2. Total sleep loss did not alter significantly the post-S_2 averaged respiratory response.

TABLE II

MEAN AND ONE STANDARD DEVIATION OF REACTION TIME (MSEC). ($N = 7$.

	Sleep deprivation	
	REM	SWS
Work-paced trials		
B	210 ± 42	209 ± 33
StD3	203 ± 32	231 ± 36
TD	223 ± 80	234 ± 28
R	190 ± 51	199 ± 29
Self-paced trials		
B	216 ± 41	227 ± 50
StD3	198 ± 32	222 ± 40
TD	217 ± 39	220 ± 34
R	189 ± 57	207 ± 26

See Table I for abbreviations.

Table II shows the mean reaction time to S_2. There were no significant differences in mean reaction times for those trials used for computing CNVs.

A post-CNV questionnaire showed that SWS deprivation did not produce significant changes in self-rating. After REM deprivation the subjects reported that they felt more tired than after SWS deprivation. The REM-deprived subjects tended to feel that the CNV task became more demanding, and that they had to exert greater effort. The night of total sleep loss following REM or SWS deprivation produced significant changes in self-ratings: the subjects felt that CNV trials were more "demanding of attention", that they had to spend more efforts in performing the task, and they felt more fatigued than during the baseline days. The subjects felt also that their reaction times were not up to par.

DISCUSSION

The present study confirms the previously published finding by Naitoh *et al.* (1971) that total sleep loss of one night attenuated the CNV magnitude and also confirms the results of studies on cardiac correlates of the CNV and CNV-like paradigms by Connor and Lang (1969), Coquery and Lacey (unpublished data), Lacey and Lacey (1970), and Papakostopoulos and McCallum (1973 *this volume*). The cardiac correlates of the present study were almost identical with those observed by Coquery and Lacey, despite the differences in types of *Ss* used (college students *vs.* Navy recruits), method of analysis (median heart rate response on beat-by-beat basis *vs.* mean heart rate response on time basis), and the nature of S_1 (a simple warning stimulus *vs.* discriminated warning stimuli). The cardiac correlates of the CNV, especially of phasic bradycardia in anticipation of S2, were not affected by REM, SWS and total sleep deprivations. Perhaps the cardiac correlates of the CNV reflected not a degree of sustained attention, but rather a subjectively experienced task demand and the willing-

ness to exert more effort in order to compensate for the detrimental effects of sleep debit. Multiple correlations of 0.630 (for the baseline average data), 0.878 (for the recovery average data), 0.823 (for StD3) and 0.863 (for TD) were obtained among the larger phasic bradycardia, the subjects' rating of exerting more effort, and that of feeling more task demands.

The absence of reliable respiratory responses indicated that the observed cardiac responses during the S_1–S_2 period were not secondary to respiratory changes.

In the present study, the differences observed during the CNV session between REM- and SWS-deprived subjects were only suggestive: the REM-deprived *Ss* showed more deleterious effects on their subjective feelings and their CNV magnitudes were more reduced than those who were deprived of SWS. Judging from the amount of REM rebound and SWS increase in the recovery sleep period, however, both REM and SWS deprivations were effectively achieved. Dement (1970) has pointed out that REM deprivation does not constitute adequate deprivation of a physiological state. Perhaps the same statement can also be made for SWS deprivation, insofar as judged by the CNV data.

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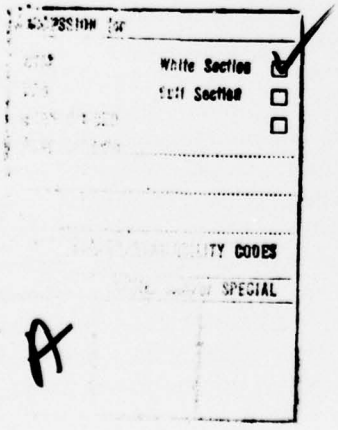

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13. ABSTRACT (1)

The contingent negative variation (CNV) from the vertex was recorded, together with autonomic variables, vertical eye movements, and reaction time (RT), from two groups of 7 Naval trainees each. The subjects received the first stimulus (S1) which was either a "high" or "low" pitched tone, 4 sec before the second "low" pitched tone (S2). With the high pitched S1, the subjects did not terminate the S2. With the low pitched S1, they were to terminate the S2 quickly as possible by closing a hand-held switch. In half of the trials, the experimenter determined the rate of executing the RT task (a work-paced condition); in the remaining half, the subject performed the RT task at their own preferred pace (self-pacing).

On each subject data were obtained on 4 baseline days, the day after 3 nights of the sleep stage deprivation, the day after one night of total sleep deprivation, and finally 2 days following recovery sleep. For the one group, sleep stage REM (rapid eye movement) was withheld from nocturnal sleep. For another, the slow wave sleep (SWS) was denied.

All subjects developed a stable discriminated CNV as well as an anticipatory heart rate deceleration to the S2 in the baseline sessions. Both REM and SWS deprivation reduced the CNV magnitudes under the self-paced condition. One night of total sleep loss following 3 nights of sleep deprivation attenuated the CNV further. Anticipatory heart rate decrease to the S2 was not affected by REM, SWS or total sleep deprivation.

Results support the hypothesis that the CNV, but not the autonomic correlates, could be used as a reliable and sensitive measure of an altered brain capacity to maintain an attentive state.

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Total sleep loss						
Work-demand						

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